

WEST Search History

LATE: Tuesday, October 12, 2010

<u>Set Name</u> side by side	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u> result set
<i>DB USPT,PGPB,JPAB,EPAB,DWPT,PLUR YES; OP ADJ</i>			
L3	L2 same 11	9	L3
L2	vaccine	35885	L2
L1	CETP	265	L1

END OF SEARCH HISTORY

FILE 'HMM' ENTERED AT 11:43:18.0000000000000000

FILE 'MELINE, CRUCEFLIT, EMBANE, BISTENHIS, BISTIA, TAILIN' ENTERED AT
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12 13 10P REM 11 12 10P REMOVED
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14 15 12 3 DETI
15 16 10P REMOVED OF REMOVED
16 17 12 3 DETI AND 14
17 18 10P REM 12 13 10P REMOVED

IN ANSWER TO OF 17 JANUARY 1997
 AN 1997:04 17 JANUARY
 IN 1997:04 17
 TI Plasmid-based **vaccine** for treating atherosclerosis
 IN Thomas, Lawrence J.
 PA T Cell Sciences, Inc., USA; Thomas, Lawrence J.
 CO 1ST Int. Appl., 48 pp.
 COIN: PLYXII

IT Patent
 LA English

PATENT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 97/1207	A1	1997:01:08	WO 1997-057094	1997:03:01
W: AL, AM, AN, AO, AP, AQ, AR, AS, AT, AU, AV, AW, AX, AY, AZ, BA, BB, BC, BD, BE, BF, BG, BH, BI, BJ, BK, BL, BM, BN, BO, BP, BQ, BR, BS, BT, BU, BV, BW, BX, BY, BZ, CA, CB, CC, CD, CE, CF, CG, CH, CI, CJ, CK, CL, CM, CN, CO, CP, CQ, CR, CS, CT, CU, CV, CW, CX, CY, CZ, DA, DB, DC, DD, DE, DF, DG, DH, DI, DJ, DK, DL, DM, DN, DO, DP, DQ, DR, DS, DT, DU, DV, DW, DX, DY, DZ, EA, EB, EC, ED, EE, EF, EG, EH, EI, EJ, EK, EL, EM, EN, EO, EP, EQ, ER, ES, ET, EU, EV, EW, EX, EY, EZ, FA, FB, FC, FD, FE, FF, FG, FH, FI, FJ, FK, FL, FM, FN, FO, FP, FQ, FR, FS, FT, FU, FV, FW, FX, FY, FZ, GA, GB, GC, GD, GE, GF, GH, GI, GJ, GK, GL, GM, GN, GO, GP, GQ, GR, GS, GT, GU, GV, GW, GX, GY, GZ, HA, HB, HC, HD, HE, HF, HG, HH, HI, HJ, HK, HL, HM, HN, HO, HP, HQ, HR, HS, HT, HU, HV, HW, HX, HY, HZ, IA, IB, IC, ID, IE, IF, IG, IH, II, IJ, IK, IL, IM, IN, IO, IP, IQ, IR, IS, IT, IU, IV, IW, IX, IY, IZ, JA, JB, JC, JD, JE, JF, JG, JH, JI, JJ, JK, JL, JM, JN, JO, JP, JQ, JR, JS, JT, JU, JV, JW, JX, JY, JZ, KA, KB, KC, KD, KE, KF, KG, KH, KI, KJ, KK, KL, KM, KN, KO, KP, KQ, KR, KS, KT, KU, KV, KW, KX, KY, KZ, LA, LB, LC, LD, LE, LF, LG, LH, LI, LJ, LK, LL, LM, LN, LO, LP, LQ, LR, LS, LT, LU, LV, LW, LX, LY, LZ, MA, MB, MC, MD, ME, MF, MG, MH, MI, MJ, MK, ML, MM, MN, MO, MP, MQ, MR, MS, MT, MU, MV, MW, MX, MY, MZ, NA, NB, NC, ND, NE, NF, NG, NH, NI, NJ, NK, NL, NM, NN, NO, NP, NQ, NR, NS, NT, NU, NV, NW, NX, NY, NZ, OA, OB, OC, OD, OE, OF, OG, OH, OI, OJ, OK, OL, OM, ON, OO, OP, OQ, OR, OS, OT, OU, OV, OW, OX, OY, OZ, PA, PB, PC, PD, PE, PF, PG, PH, PI, PJ, PK, PL, PM, PN, PO, PP, PQ, PR, PS, PT, PU, PV, PW, PX, PY, PZ, QA, QB, QC, QD, QE, QF, QG, QH, QI, QJ, QK, QL, QM, QN, QO, QP, QQ, QR, QS, QT, QU, QV, QW, QX, QY, QZ, RA, RB, RC, RD, RE, RF, RG, RH, RI, RJ, RK, RL, RM, RN, RO, RP, RQ, RR, RS, RT, RU, RV, RW, RX, RY, RZ, SA, SB, SC, SD, SE, SF, SG, SH, SI, SJ, SK, SL, SM, SN, SO, SP, SQ, SR, SS, ST, SU, SV, SW, SX, SY, SZ, TA, TB, TC, TD, TE, TF, TG, TH, TI, TJ, TK, TL, TM, TN, TO, TP, TQ, TR, TS, TU, TV, TW, TX, TY, TZ, UA, UB, UC, UD, UE, UF, UG, UH, UI, UJ, UK, UL, UM, UN, UO, UP, UQ, UR, US, UT, UU, UV, UW, UX, UY, UZ, VA, VB, VC, VD, VE, VF, VG, VH, VI, VJ, VK, VL, VM, VN, VO, VP, VQ, VR, VS, VT, VU, VV, VW, VX, VY, VZ, WA, WB, WC, WD, WE, WF, WG, WH, WI, WJ, WK, WL, WM, WN, WO, WP, WQ, WR, WS, WT, WU, WV, WW, WX, WY, WZ, XA, XB, XC, XD, XE, XF, XG, XH, XI, XJ, XK, XL, XM, XN, XO, XP, XQ, XR, XS, XT, XU, XV, XW, XX, XY, XZ, YA, YB, YC, YD, YE, YF, YG, YH, YI, YJ, YK, YL, YM, YN, YO, YP, YQ, YR, YS, YT, YU, YV, YW, YX, YY, YZ, ZA, ZB, ZC, ZD, ZE, ZF, ZG, ZH, ZI, ZJ, ZK, ZL, ZM, ZN, ZO, ZP, ZQ, ZR, ZS, ZT, ZU, ZV, ZW, ZX, ZY, ZZ				
CA 2284424	AA	1997:11:08	CA 1997-2284424	1997:11:01
AU 9029946	A1	1997:11:08	AU 1997-2284424	1997:11:01
US 721729	B2	1997:11:08		
EP 0144307	A1	1997:06:12	EP 1997-024849	1997:06:01
E: AT, BE, CH, DE, DK, ES, FF, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2000-000000	TE	2000:01:08	JP 1997-000000	1997:01:01
US 6194031	B1	1997:01:08	US 1997-000000	1997:01:01
US 1997-000000	A	1997:01:08		
US 1997-000000	AD	1997:01:08		
US 1997-000000	B	1997:01:08		
US 1997-000000	C	1997:01:08		
AB A plasmid-based vaccine is provided that is based on the combination of DNA segments coding for one or more E cell epitopes of CETP and one or more broad range helper T cell epitopes. Administration of the plasmids as a vaccine to a vertebrate subject provides an immune response to the subject's endogenous CETP and modulation of CETP activity, leading to prevention or reversal of various manifestations of heart disease. The vaccines provide an advantageous strategy for the prevention or treatment of atherosclerosis.				

AN 1987-10-16 ELECTRONIC
TI INA plasmid based vaccine;
nucleic acid vaccine for cardiovascular disease

AN Thomas L J
PA T-cell-sci.
LC Needham, MA, USA.
FI WO 840127, May 1985
AI NO 84-7-00124, May 1985
PBAI US 4,487,817, 21 Feb 1987; US 4,487,818, 21 May 1987
IT Patent
LA English
CS WPI: 1987-049781 (B)
AB

A new nucleic acid **vaccine** comprises a INA sequence I encoding an immunogenic protein, where at least 1 segment of I encodes a B-lymphocyte epitope of cholesteryl ester-transferase protein **CETP** linked with at least 1 segment encoding a broad range helper T-lymphocyte epitope, where the nucleotide segment is operably linked to a promoter for directing transcription of I in a mammalian cell. Also claimed are: a INA based plasmid **vaccine** comprising a nucleotide sequence comprising the immediate early promoter/enhancer region of cytomegalic virus operably linked to a structural INA segment encoding an immunogenic protein selected from polypeptide regions of a disclosed protein sequence; a INA plasmid based **vaccine** comprising a INA segment encoding a broad range T-lymphocyte epitope. The nucleic acid **vaccine** can be used to elevate the ratio of circulating high density lipoproteins to circulating low density lipoproteins, very low density lipoproteins or total cholesterol in a human and for reducing the level of endogenous **CETP** activity in a human. The **vaccine** can also be used to induce antibodies and for cardiovascular disease therapy. 6pp

ANOTHER NAME: EMBASE; EMBASE; EMBASE; EMBASE; EMBASE;
 AB: EMBASE; EMBASE;
 TI: Genetic polymorphisms and activity of cholesteryl ester transfer protein:
 CETP : Should we be measuring them?
 AU: Ordoñez J.M.
 AS: J.M. Ordoñez, Lipid Metabolism Laboratory, Jean Mayer USDA Hum. Nutr. Res.
 Ctr., Tufts University, Boston, MA, United States. jdores@hs.tufts.edu
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 Boston, MA 02115, USA.
 ISN: 1484-0021 ISSN: 1484-0021
 CY: Germany
 JT: Journal; Article
 FS: 118 Cardiovascular Diseases and Cardiovascular Surgery
 122 Human Genetics
 137 Clinical Biochemistry
 138 Drug Literature Index
 LA: English
 SL: English
 AB: Cholesteryl ester transfer protein. CETP is a plasma
 glycoprotein that mediates the transfer of cholesteryl ester from high
 density lipoproteins HDL to triglyceride-rich lipoproteins in exchange
 for triglycerides. Several approaches are currently being used in research
 laboratories to measure its activity and or mass. However, these assays
 are not standardized and it is not possible to compare data from different
 laboratories. Also, we lack enough information to assess the value of this
 variable as a coronary heart disease (CHD) predictor. Several genetic
 variants at CETP locus have been identified and they have been
 generally associated with increased HDL-cholesterol concentrations.
 However, there is no consensus about the association of this CETP
 -related increase in HDL-cholesterol and protection against CHD.
 Nevertheless, the most recent evidence from the common CETP
 -Taql-B polymorphism shows that the lower CETP activity
 associated with the presence of this polymorphism decreases CHD risk in
 men. Based on this and previous evidence, there has been an interest in
 the development of CETP inhibitors as a tool to increase
 HDL-cholesterol, thus reducing CHD risk. However, it should be noted that
 the evidence about the cardioprotective role of these drugs is not yet
 available.

AN 000004 1F 1- MEDLINE
 AM 1- 1111 - MEDLINE
 IN 1-46174 PubMed ID: 11111111
 TI Vaccine induced antibodies inhibit CETP activity in vivo and reduce atherosclerosis in a rabbit model of atherosclerosis.
 JN Journal of Atherosclerosis Thrombosis Vasc Biol. 2009 Sep; 11(9):1111-1118.
 AU Bittershaug JW; Miller LG; Thomas LJ; Hwang ML; Herold JM; Emmett M L; Isley CL; Adams R; Hammond EA; Beattie L L; Wilson AJ; Marsh H D; Ryan TJ
 AD ARSIT Immunotherapeutics, Inc., Needham, MA 01944, USA.
 DA bittershaug@arstitimmune.com
 NC HL-11111 NLMID
 NO ATHEROSCLEROSIS, THROMBOSIS, AND VASCULAR BIOLOGY, 11(9): 1111-1118, 2009.
 JO Journal code: JA 1111. ISSN: 1524-4033.
 CY United States
 IT Journal; Article; JOURNAL ARTICLE
 LA English
 ES Priority Journals
 EM 1111
 EI Entered STN: 11111111
 LU Last Updated on STN: 11111111
 ER Entered Medline: 11111111
 AB Using a vaccine approach, we immunized New Zealand White rabbits with a peptide containing a region of cholesteryl ester transfer protein (CETP) known to be required for neutral lipid transfer function. These rabbits had significantly reduced plasma CETP activity and an altered lipoprotein profile. In a cholesterol-fed rabbit model of atherosclerosis, the fraction of plasma cholesterol in HDL was 42% higher and the fraction of plasma cholesterol in LDL was 14% lower in the CETP-vaccinated group than in the control-vaccinated group. Moreover, the percentage of the aorta surface exhibiting atherosclerotic lesion was 19.6% smaller in the CETP-vaccinated rabbits than in controls. The data reported here demonstrate that CETP activity can be reduced in vivo by vaccination with a peptide derived from CETP and support the concept that inhibition of CETP activity in vivo can be antiatherogenic. In addition, these studies suggest that vaccination against a self-antigen is a viable therapeutic strategy for disease management.

the 1990s, the number of people in the world who are illiterate has declined by 100 million. The number of people who are illiterate is still 1 billion, but the number of people who are literate is 1.7 billion. The number of people who are illiterate is still 1 billion, but the number of people who are literate is 1.7 billion. The number of people who are illiterate is still 1 billion, but the number of people who are literate is 1.7 billion.

[illegible]

11. An innovative device approach for the treatment of low plasma
HIV-1 viral loads.

1. *Chlorophyll a* and *Chlorophyll b* were determined by the method of Arar and Collins (1971).

[illegible]

Table 1. *Continued*

[illegible]

1997, 1998, 1999, 2000, 2001, 2002, 2003, 2004, 2005, 2006, 2007, 2008, 2009, 2010, 2011, 2012, 2013, 2014, 2015, 2016, 2017, 2018, 2019, 2020, 2021, 2022, 2023, 2024, 2025, 2026, 2027, 2028, 2029, 2030, 2031, 2032, 2033, 2034, 2035, 2036, 2037, 2038, 2039, 2040, 2041, 2042, 2043, 2044, 2045, 2046, 2047, 2048, 2049, 2050, 2051, 2052, 2053, 2054, 2055, 2056, 2057, 2058, 2059, 2060, 2061, 2062, 2063, 2064, 2065, 2066, 2067, 2068, 2069, 2070, 2071, 2072, 2073, 2074, 2075, 2076, 2077, 2078, 2079, 2080, 2081, 2082, 2083, 2084, 2085, 2086, 2087, 2088, 2089, 2090, 2091, 2092, 2093, 2094, 2095, 2096, 2097, 2098, 2099, 2100, 2101, 2102, 2103, 2104, 2105, 2106, 2107, 2108, 2109, 2110, 2111, 2112, 2113, 2114, 2115, 2116, 2117, 2118, 2119, 2120, 2121, 2122, 2123, 2124, 2125, 2126, 2127, 2128, 2129, 2130, 2131, 2132, 2133, 2134, 2135, 2136, 2137, 2138, 2139, 2140, 2141, 2142, 2143, 2144, 2145, 2146, 2147, 2148, 2149, 2150, 2151, 2152, 2153, 2154, 2155, 2156, 2157, 2158, 2159, 2160, 2161, 2162, 2163, 2164, 2165, 2166, 2167, 2168, 2169, 2170, 2171, 2172, 2173, 2174, 2175, 2176, 2177, 2178, 2179, 2180, 2181, 2182, 2183, 2184, 2185, 2186, 2187, 2188, 2189, 2190, 2191, 2192, 2193, 2194, 2195, 2196, 2197, 2198, 2199, 2200, 2201, 2202, 2203, 2204, 2205, 2206, 2207, 2208, 2209, 2210, 2211, 2212, 2213, 2214, 2215, 2216, 2217, 2218, 2219, 2220, 2221, 2222, 2223, 2224, 2225, 2226, 2227, 2228, 2229, 2230, 2231, 2232, 2233, 2234, 2235, 2236, 2237, 2238, 2239, 2240, 2241, 2242, 2243, 2244, 2245, 2246, 2247, 2248, 2249, 2250, 2251, 2252, 2253, 2254, 2255, 2256, 2257, 2258, 2259, 2260, 2261, 2262, 2263, 2264, 2265, 2266, 2267, 2268, 2269, 2270, 2271, 2272, 2273, 2274, 2275, 2276, 2277, 2278, 2279, 2280, 2281, 2282, 2283, 2284, 2285, 2286, 2287, 2288, 2289, 2290, 2291, 2292, 2293, 2294, 2295, 2296, 2297, 2298, 2299, 2300, 2301, 2302, 2303, 2304, 2305, 2306, 2307, 2308, 2309, 2310, 2311, 2312, 2313, 2314, 2315, 2316, 2317, 2318, 2319, 2320, 2321, 2322, 2323, 2324, 2325, 2326, 2327, 2328, 2329, 2330, 2331, 2332, 2333, 2334, 2335, 2336, 2337, 2338, 2339, 2340, 2341, 2342, 2343, 2344, 2345, 2346, 2347, 2348, 2349, 2350, 2351, 2352, 2353, 2354, 2355, 2356, 2357, 2358, 2359, 2360, 2361, 2362, 2363, 2364, 2365, 2366, 2367, 2368, 2369, 2370, 2371, 2372, 2373, 2374, 2375, 2376, 2377, 2378, 2379, 2380, 2381, 2382, 2383, 2384, 2385, 2386, 2387, 2388, 2389, 2390, 2391, 2392, 2393, 2394, 2395, 2396, 2397, 2398, 2399, 2400, 2401, 2402, 2403, 2404, 2405, 2406, 2407, 2408, 2409, 2410, 2411, 2412, 2413, 2414, 2415, 2416, 2417, 2418, 2419, 2420, 2421, 2422, 2423, 2424, 2425, 2426, 2427, 2428, 2429, 2430, 2431, 2432, 2433, 2434, 2435, 2436, 2437, 2438, 2439, 2440, 2441, 2442, 2443, 2444, 2445, 2446, 2447, 2448, 2449, 2450, 2451, 2452, 2453, 2454, 2455, 2456, 2457, 2458, 2459, 2460, 2461, 2462, 2463, 2464, 2465, 2466, 2467, 2468, 2469, 2470, 2471, 2472, 2473, 2474, 2475, 2476, 2477, 2478, 2479, 2480, 2481, 2482, 2483, 2484, 2485, 2486, 2487, 2488, 2489, 2490, 2491, 2492, 2493, 2494, 2495, 2496, 2497, 2498, 2499, 2500, 2501, 2502, 2503, 2504, 2505, 2506, 2507, 2508, 2509, 2510, 2511, 2512, 2513, 2514, 2515, 2516, 2517, 2518, 2519, 2520, 2521, 2522, 2523, 2524, 2525, 2526, 2527, 2528, 2529, 2530, 2531, 2532, 2533, 2534, 2535, 2536, 2537, 2538, 2539, 2540, 2541, 2542, 2543, 2544, 2545, 2546, 2547, 2548, 2549, 2550, 2551, 2552, 2553, 2554, 2555, 2556, 2557, 2558, 2559, 2560, 2561, 2562, 2563, 2564, 2565, 2566, 2567, 2568, 2569, 2570, 2571, 2572, 2573, 2574, 2575, 2576, 2577, 2578, 2579, 2580, 2581, 2582, 2583, 2584, 2585, 2586, 2587, 2588, 2589, 2590, 2591, 2592, 2593, 2594, 2595, 2596, 2597, 2598, 2599, 2600, 2601, 2602, 2603, 2604, 2605, 2606, 2607, 2608, 2609, 2610, 2611, 2612, 2613, 2614, 2615, 2616, 2617, 2618, 2619, 2620, 2621, 2622, 2623, 2624, 2625, 2626, 2627, 2628, 2629, 2630, 2631, 2632, 2633, 2634, 2635, 2636, 2637, 2638, 2639, 2640, 2641, 2642, 2643, 2644, 2645, 2646, 2647, 2648, 2649, 2650, 2651, 2652, 2653, 2654, 2655, 2656, 2657, 2658, 2659, 2660, 2661, 2662, 2663, 2664, 2665, 2666, 2667, 2668, 2669, 2670, 2671, 2672, 2673, 2674, 2675, 2676, 2677, 2678, 26

Environ Biol Fish (2015) 98:1011–1020

One determinant of plasma HDL-cholesterol concentration is cholesterol ester transfer protein (CETP) activity. Inhibition of CETP activity increases plasma HDL-C, thus providing a potential therapeutic target for the treatment of atherosclerosis. Using a vaccine approach, we immunized New Zealand White rabbits with a peptide containing a region of CETP known to be required for neutral lipid transfer function. CETP-vaccinated rabbits had significantly reduced plasma CETP activity and an altered lipoprotein profile compared with control rabbits. In a cholesterol-fed rabbit model of atherosclerosis, the fraction of plasma HDL-cholesterol in HDL was 42% higher, and the fraction of plasma cholesterol in LDL was 24% lower in the CETP-vaccinated group compared with the control-vaccinated group. Moreover, the percentage of the aorta surface exhibiting atherosclerotic lesion was 38.6% smaller in the CETP-vaccinated rabbits compared with controls. The data reported here demonstrate that CETP activity can be reduced in vivo by vaccination with a peptide derived from CETP, and support the concept that inhibition of CETP activity in vivo can be anti-atherogenic. Currently, this vaccine is in clinical trials.

1. The first part of the document is a list of references. The references are listed in a standard format, with the author's name, the title of the work, and the publisher. The references are as follows:

1. The first part of the document is a list of references. The references are listed in a standard format, with the author's name, the title of the work, and the publisher. The references are as follows:

10 ANSWER: **ARTHEROSCLEROSIS** **ARTHEROSCLEROSIS** **ARTHEROSCLEROSIS** **ARTHEROSCLEROSIS** **ARTHEROSCLEROSIS**
 20 **ARTHEROSCLEROSIS**
 30 Cholesteryl ester transfer protein corrects dysfunctional high density
 40 lipoproteins and reduces aortic atherosclerosis in lecithin:cholesterol
 50 acyltransferase transgenic mice;
 60 adenovirus vector-mediated human phosphatidylcholine:sterol-O-
 70 acyltransferase expression in mice for **arterosclerosis**
 80 **model**
 90 Foster B; Chase M; Amar M C; Williams B L; Shanbhogue P L; Sanyal B;
 100 Fruchart-Kagik J; Bain J A; Koch J A; Hays R F; Brewer T H B;
 110 *Santamarina-Fojo S
 120 Nat.Inst.Health-Bethesda; Jackson-Lab-Bar-Harbor; INSERM
 130 National Institutes of Health, Molecular Disease Branch, NHLBI, Building
 140 1, Room 2N111, 1 Center Drive MSC 1035, Bethesda, MD 20814-6088, USA.
 150 J.Biol.Chem.; 1998; 273, 22, 14111-1
 160 CCEW: 002141 ISSN: 0021-9738
 170 Journal
 180 English
 190 Human phosphatidylcholine:sterol-O-acyltransferase (PCAT, EC-2.3.1.43)
 200 expression adenovirus vector in mice (PCAT-Tg) leads to increased high
 210 density lipoprotein (HDL) cholesterol levels but paradoxically, enhanced
 220 atherosclerosis. PCAT-Tg were cross bred with cholesteryl ester transfer
 230 protein (CETP) -Tg mice to test the hypothesis that the absence
 240 of CETP in PCAT-Tg mice facilitates the accumulation of
 250 dysfunctional HDL leading to impaired reverse cholesterol transport and
 260 the development of a pro-atherogenic state. Expression of CETP
 270 in PCAT-Tg mice reduced total cholesterol, reflecting a decrease in HDL
 280 cholesterol levels. CETP normalized both the plasma clearance
 290 of 3H cholesteryl esters (3H CE) from HDL as well as the liver uptake
 300 of 3H CE from HDL in PCAT-Tg mice. CETP expression reduces
 310 atherosclerosis in PCAT-Tg mice by restoring the functional properties of
 320 PCAT-Tg mouse HDL and promoting the hepatic uptake of HDL-cholesteryl
 330 ester. Therefore CETP expression is beneficial in
 340 pro-atherogenic states that result from impaired reverse cholesterol.
 350 **AB**

AB ANSWER L 1F 1-1 ENHANCE Copyright © 1994 Elsevier B.V. All rights reserved.
AN 1 1F 1-1 ENHANCE
TI Molecular mechanisms, lipoprotein abnormalities and atherogenicity of
hyperalphalipoproteinemia.
AU Tanashita S.; Maruyama T.; Hirano H.; Sakai N.; Nakajima H.; Matsuzawa Y.
CO S. Tanashita, Department of Internal Medicine, Graduate School of
Medicine, Osaka University, 1-1 Yamadaoka, Suita, Osaka 565-0871, Japan.
shimazumi.med.osaka-u.ac.jp
SO Atherosclerosis, 1 1994, 101, 1-181.

FOIS: 1-1
ISSN: 0921-2181 CODEN: ATHSHL
FOL 3 111-115 1994
CY Ireland
IT Journal; General Review

FS 117 Public Health, Social Medicine and Epidemiology
117 Cardiovascular Diseases and Cardiovascular Surgery
122 Human Genetics
123 Hematology
123 Clinical Biochemistry
123 General Pathology and Pathological Anatomy

LA English
SL English

AB Hyperalphalipoproteinemia (HALP) is caused by a variety of genetic and environmental factors. Among these, plasma cholesteryl ester transfer protein (CETP) deficiency is the most important and frequent cause of HALP in the Asian populations. CETP facilitates the transfer of cholesteryl ester (CE) from high density lipoprotein (HDL) to apolipoprotein (apo) B-containing lipoproteins, and is a key protein in the reverse cholesterol transport system. The deficiency of CETP causes various abnormalities in the concentration, composition, and function of both HDL and low density lipoprotein (LDL). The significance of CETP in terms of atherosclerosis had been controversial. However, the in vitro evidence showed large CE-rich HDL particles in CETP deficiency are defective in cholesterol efflux. Similarly, scavenger receptor (SR) B1 (SR-B1) knockout mice show a marked increase in HDL-cholesterol but accelerated atherosclerosis in atherosclerosis-susceptible mice. Recent epidemiological studies in Japanese-Americans and in Omagari area where HALP subjects with the intron 14 splicing defect of CETP gene are markedly frequent, have demonstrated an increased incidence of coronary atherosclerosis in CETP deficient patients. Thus, CETP deficiency is a state of impaired reverse cholesterol transport which may possibly lead to the development of atherosclerosis. The current review will focus on the molecular mechanisms and atherogenicity of HALP, especially CETP deficiency. Copyright © 1994 Elsevier Science Ireland Ltd.

BT ABSTRACTS OF THE AMERICAN JOURNAL OF MEDICAL SCIENCES
AB 117414 ABSTRACT
TI Cholesteryl ester transfer protein inhibitors.
AU Shinkai H.
SO H. Shinkai, Central Pharmaceutical Res. Inst., JT Inc., 1-1 Morinoshi-cho,
Takahara, Osaka 595-0051, Japan. hshinkai@phs.jti.co.jp
SO Expert Opinion on Therapeutic Patents, 11(5) 735-745.
Pis: 4
ISSN: 1364-8774 CODEN: EOTPHS
JO United Kingdom
JT Journal; General Review
FS 719 Cardiovascular Diseases and Cardiovascular Surgery
FS 787 Drug Literature Index
LA English
SI English
AB As well as hypercholesterolemia, low levels of high-density lipoprotein cholesterol (HDL-C) are critical risk factors for atherosclerosis and coronary heart disease (CHD). Although fibrate, simvastatin and niacin can be used for the treatment of a low HDL-C level, their effects, however, are not wholly satisfactory. Thus, better drugs for the elevation of HDL-C are desired. Among the many methods that may be used to raise HDL-C levels, this review focuses on small molecule inhibitors of cholesteryl ester transfer protein (CETP) and summarizes recent patent and journal data.